Clinical Study of Thirteen Patients with Spinal Cord Infarction

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Background: A concept of sensory tracts in the spinal cord has been established in relation to a dorsolateral pathway which is located in the posterior part of the lateral column and conveys the deep sense. *Methods:* The clinical status at onset, neurological symptoms, and magnetic resonance imaging (MRI) findings in 13 patients of spinal cord infarction were studied. *Results:* The clinical status was acute in 11 patients and subacute in 2 patients. Palsy of the extremities was noted in 11 patients. Segmental sensory disturbance was shown in all patients. One patient showed disturbance of all senses and paraplegia, which indicated transverse myelopathy. In the other 12 patients, 11 patients showed impairment of pain sense although joint position sense was preserved, excluding 1 patient whose sensory disturbance showed dysesthesia alone. In these 11 patients, soft touch and vibration senses were impaired in 7 patients. Abnormality of spinal cord MRI was detected 7 patients. The lesions were located in the cervical cord in 3 patients, cervical to thoracic cord in 1 patient, and thoracic cord in 3 patients. *Conclusions:* In the 11 patients in whom pain sense was impaired and joint position sense was preserved, involvement of the anterior spinal cord artery (ASCA) was the mainstay. Impairment of vibration sense was accompanied in 7 patients in patients of ASCA infarction. It was speculated that impairment of vibration sense can occur in patients with ASCA infarction whose ischemia spread to the dorsolateral pathway in the posterior part of the lateral column.

Key Words: Spinal cord infarction—anterior spinal cord artery—lateral column—dorsolateral pathway—vibration sense

The spinal cord is supplied with the anterior and posterior spinal cord arteries.[1-4] The anterior spinal cord artery (ASCA) supplies the anterior and lateral columns,[1-4] and the posterior spinal cord artery (PSCA) supplies the posterior column.[1,2,4] Spinal cord infarction (SCI) is usually divided into 2 forms; i.e., anterior and posterior spinal cord artery infarction, based on the distribution of each arterial supply.[1,2,4] A new understanding of sensory tracts in the spinal cord has been reported recently.[5-7] In convey of information from superficial senses, the concept of the anterolateral quadrant has been put forward because tracts in the superficial senses are located within the anterior and lateral columns.[7,8] Furthermore, the presence of tracts for deep sense in the lateral column has been identified recently.[5] Here, we studied the clinical characteristics of SCI, based on the recently reported understanding of the sensory tracts in the spinal cord.[5]

Patients and Methods

Characteristics of the Enrolled Patients

Thirteen patients aged from 51 to 80 years old (11 males, 2 females) were enrolled in our study. They were admitted to the Neurology Ward in our hospital from 2002 to 2013 with complaints of myelopathic signs with acute to subacute onset. Diagnosis of SCI was made,

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when the 5 items as below were satisfied; i.e. (1) a monophasic course, (2) presence of myelopathic symptoms with acute to subacute onset, (3) other neurological diseases that can affect the spinal cord (arteriovenous malformation/fistula, hemorrhage, myelitis, demyelinating disease, and sarcoidosis) were negligible, based on the clinical course, magnetic resonance imaging (MRI) findings of the spinal cord, and laboratory data, (4) presence of correlation between the neurological symptoms and locations of the lesions in the spinal cord, (5) The 4 items of (1) to (4) were satisfied whereas no lesions were detected in the spinal cord. We reviewed their clinical data and initial symptoms at onset, neurological symptoms on admission, vascular risk factors, other complications, and MRI findings of the spinal cord in each patient.

Results

Background of Enrolled Patients, Vascular Risk Factors, and Other Complications

The age of enrolled patients ranged from 51 to 81 years old (Table 1). Vascular risk factors were noted in all patients. Hypertension was most frequent (11 patients), followed by dyslipidemia in 5 patients, and diabetes mellitus was found in 3 patients. Two patients were current smokers. Complications with aortic aneurysm were enrolled in 4 patients. SCI occurred at the chronic stage after operation for aortic aneurysm in 2 patients (patients 3, 10). In 2 patients, operation for abdominal aortic aneurysm was performed 2 years before onset in 1 patient (patient 3) and at 10 years before onset in another patient (patient 10). In contrast, the other 2 patients suffered from SCI in an acute stage of operation for aortic aneurysm (patients 1, 13). SCI occurred simultaneously with the onset of dissecting aneurysm of the ascending aorta to the common iliac artery in 1 patient (patient 1). Operation for dissecting thoracic aortic aneurysm was performed in 1 patient at 1 day before onset (patient 13).

Clinical Status and Initial Symptoms

Clinical status at onset was acute-onset in 11 patients and subacute-onset in 2 patients (Table 1). The first complaints were motor weakness in the extremities in 11 patients, numbness/pain of the extremities in 5 patients, and back pain in 5 patients.

Neurological Symptoms on Admission

Palsy of the extremities was noted in 11 patients (Table 2). The type of palsy of the extremities was paraplegia in 6 patients (patients 1, 4, 7, 9, 10, 12), tetraplegia in 2 patients (patients 2, 5), and monoplegia in 2 patients (left upper extremity; 1 patient [patient 6], left lower extremity; 1 patient [patient 13]). Palsy of the bilateral

Table 1. *Breakdown of 13 patients of spinal cord infarction*

No.	Age/sex	Clinical form at onset	Initial symptoms			Vascular risk factors	Other complications
			Back pain	Motor weakness	Numbness/pain		
1	62/m	Acute	+	+ (bil. legs)	−	HTN, SM	Dissecting aortic aneurysm (ascending aorta to common iliac artery)
2	51/f	Acute	+	+ (lt leg)	+ (four ext.)	HTN, DL	−
3	63/m	Acute	−	−	+ (bil. legs)	HTN	Operation for dissecting AAA (2 y before onset of infarction)
4	80/m	Acute	−	+ (bil. legs)	−	HTN	−
5	57/m	Acute	−	+ (four ext.)	+ (bil. arms)	HTN	−
6	52/m	Acute	−	+ (lt arm)	−	HTN, DM	−
7	70/m	Acute	+	+ (bil. legs)	+ (bil. legs)	DL, DM	−
8	51/m	Acute	+	−	+ (bil. arms)	HTN, DM, SM	−
9	67/m	Acute	−	+ (bil. legs)	−	HTN, DL	−
10	68/m	Subacute	−	+ (bil. legs)	−	HTN	Operation for dissecting AAA (10 y before onset of infarction)
11	71/m	Acute	−	+ (lt leg)	−	HTN, DL	−
12	53/f	Subacute	+	+ (bil. legs)	−	DL	−
13	67/m	Acute	−	+ (lt arm, leg)	−	HTN	Operation of dissecting thoracic aortic aneurysm (1 d before onset of infarction)

Abbreviations: AAA, abdominal aortic aneurysm; bil, bilateral; DL, dyslipidemia; DM, diabetes mellitus; ext, extremeities; f, female; HTN, hypertension; lt, left; m, male; No, number; SM, smoking.

upper extremities was noted in 1 patient (patient 8). The severity of palsy of the lower extremities was moderate to severe in 7 patients (patients 1, 2, 4, 7, 9, 12, 13). Bladder and rectal dysfunction accompanied other symptoms in 7 patients. Segmental sensory disturbance was noted in all patients. The segment of the sensory disturbance was located at the level of the thoracic cord in 9 patients (patients 1, 3, 4, 6-10, 12), the cervical cord in 2 patients (patients 2, 5), and the lumbar cord in 1 patient (patient 13). The segment of the sensory disturbance in 1 patient was mapped at the thoracic cord level on the lateral side and the lumbar cord level on another side (patient 11). One patient noted total sensory disturbance (patient 1). Seven patients showed sensory disturbance in pain, soft touch, and vibration senses in the absence of impairment of joint position sense (patients 3, 7, 9-13). Among these 7 patients, anesthesia was presented in the lateral leg in 1 patient (patient 3). Four patients showed superficial sensory disturbance in the absence of deep sense (patients 2, 4-6). The sensory disturbance in 2 patients was associated with pain and soft touch senses (patients 4, 6), and 2 other patients noted sensory disturbance in pain alone (patients 2, 5). Two patients presented with dysesthesia (patients 8, 13), and of these 1 patient showed dysesthesia alone in sensory disturbance (patient 8).

MRI Findings of the Spinal Cord

An abnormal intensity area was detected in 7 patients (Tables 2, 3). MRI examinations were performed within the duration between 2 days and 1 month after onset. The abnormal high intensity area (HIA) was detected in T2-weighted image in 6 patients and in T1-weighted images in 1 patient (patient 6). Lesions were located in the cervical cord in 3 patients (patients 5, 6, 8), the cervical to thoracic cord in 1 patient (patient 2), and the thoracic cord in 3 patients (patients 9, 12, 13) in the sagittal section. The axial images were obtainable in 4 patients (patients 5, 9, 12, 13) (Table 3). The duration between the photographing time of axial images and onset was the same as that in (Table 2) in 3 patients (patients 9, 12, 13) and 2 months in 1 patient (patient 5). In the axial section of the 4 patients, lesions were located in the bilateral anterior regions in 2 patients (patients 5, 9) and the lateral column on the left side in 1 patient (patient 13). The lesion was located in the central to anterior region and was circumscribed by the faint HIA in 1 patient (patient 12).

Discussion

The anterior and posterior radicular arteries diverge into the rostral and caudal branches in the vertical direction, respectively.[1] In the anterior median fissure, the ASCA is conformed by the anastomosis of the rostral and caudal branches (Fig 1).[1,2] In the posterior aspect, the 2 PSCAs are conformed by the anastomosis of the rostral and caudal branches at the bilateral posterior lateral fissures (Fig 1).[1,2] The anterior radicular artery is more developed at the C6/7, Th9/10, and L2 levels.[1] The 2 watershed areas exist at the Th4 and L1 levels due to the disequilibrium of blood supply.[1] The ASCA bifurcates into the central artery and the coronary artery (Fig 1).[1,2] The coronary artery connects with the PSCA (Fig 1).[1,2] The spinal cord can be divided into 3 regions- medial, lateral, and dorsal-based on the distribution of each three arteries (Fig 2).[1,2] The medial region is supplied by the central artery (Fig. 1, 2). [1,2] The lateral region is supplied by the coronary artery (Fig. 1, 2);[1,2] however, the extent of the region in the distribution of the coronary artery is limited in the surface layer of the anterior and lateral columns (Fig 2). [1] The PSCA supplies the posterior column (Fig. 1, 2).[1] There are the overlapping areas among the territories of each artery. One third of the transverse section is considered to be an overlapping area.[1] With ischemia of the PSCA, the ischemic lesion could spread to the posterior part of the lateral column where the corticospinal tract exits by ischemia of the coronary artery. As a result, palsy of the extremities accompanies ischemia of the PSCA.[1]

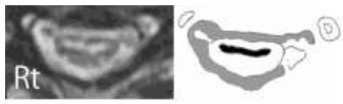
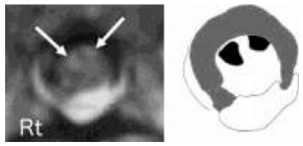
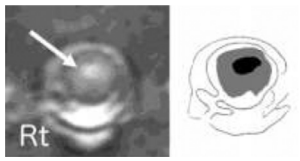
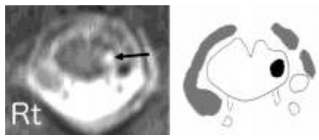
In recent years, the conveying of sensory information in the spinal cord has been reconsidered.[5,7,8] Tracts of pain and temperature sensing are comprised of the spinothalamic, spinoreticular, spinomesencephalic, and spinohypothalamic tracts (Fig 3).[7-9] These 4 tracts are located in the anterior and lateral columns, and are called the anterolateral quadrant (Fig 3).[7,8] The spinothalamic tract is composed of 2 tracts; i.e., the anterior spinothalamic tract and the lateral spinothalamic tract, and it conveys direct information on pain and temperature (Fig 3).[10,11] Functionally, these 2 tracts have been considered to be 1 pathway (Fig 4, A).[6,10] Neurofibers conveying information on pain and temperature enter the spinal cord from the dorsal horn and decussate at the same level as the entrance, and they approach the lateral and anterior spinothalamic tracts (Fig 4, A).[9] These 2 tracts then run adjacently to each other and finally approach the ventral posterolateral (VPL) nucleus in the thalamus (Fig 4, A).[5,6] Neurofibers conveying information on deep sense enter the dorsal horn (Fig 4, B).[1,5] Many of the fibers enter the posterior column on the ipsilateral side and ascend in the posterior column (Fig 4, B).[5] These fibers decussate and constitute the medial lemniscus at the medulla, then finally approach the VPL nucleus (Fig 4, B).[5] The other neurofibers of deep sense enter the ipsilateral dorsolateral pathway which is located in the posterior part of the lateral column and ascend to the lateral cervical nucleus at the C1 and C2 level, and they finally approach the VPL nucleus through the medial lemniscus after decussation in the cervical cord (Fig 4, B).[5] Neurofibers of soft touch sense bifurcate into the posterior column on the ipsilateral side and ascend in the posterior column (Fig 4, C).[5] The other neurofibers of soft touch sense ascend in either the dorsolateral pathway on the ipsilateral side or the lateral spinothalamic tract on the contralateral side (Fig 4, C).[5] Neurofibers of soft touch sense as

Table 2. *Neurological and MRI findings of 13 patients of spinal cord infarction*

No.	Neurological findings on admission									HIA in MRI	Duration between MRI/onset
	Palsy of extremities		B/R DF	Sensory disturbance (rt/lt)							
	Upper (rt/lt)	Lower (rt/lt)		Sensory level	DE	PP (pain)	ST	Vib	JPS		
1	−/−	+++/+++	+	bil. Th11	−	++/++	++/++	++/++ (legs)	+++/+++ (toes)	Normal	7 d
2	+/+	++/+++	+	bil. C4	−	++/++	−	−	−	C5 to Th3 (T2)	8 d
3	−/−	−/−	−	bil. Th10	−	+++ (AE)/+	+++ (AE)/+	+++ (AE)/++ (legs)	−	Normal	12 d
4	−/−	+++/+++	+	bil. Th6	−	+++/++	+++/++	−	−	Normal	17 d
5	++/+	+/+	+	rt C8 to L5, lt C8	−	++/++	−	−	−	C2, C4 to C6 (T2)	3 d
6	−/+	−/−	−	rt Th4 to L2 lt Th4 to Th10	−	+/+	+/+	−	−	C5 to C6 (T1)	5 d
7	−/−	+++/++	+	bil Th3	−	++/++	++/++	++/++ (distal legs)	−	Normal	5 d
8	+/+	−/−	−	bil. Th5 to Th11	+	−	−	−	−	C4 to C6 (T2)	2 d
9	−/−	+++/+++	+	bil Th8		+++/+++	++/++	++/++ (distal legs)	−	Th8 to Th10 (T2)	3 d
10	−/−	+/+	−	bil. Th10	−	++/++	++/++	+/+ (distal legs)	−	Normal	25 d
11	−/−	−/−	−	rt Th11, lt L2	−	++/++	++/++	++/+ (legs)	−	Normal	7 d
12	−/−	+++/+++	+	bil. Th5	−	+++/+++	+/+	+++/+++ (distal legs)	−	Th3 to Th10 (T2)	9 d
13	−/−	−/++	−	rt L1	+	++/− (PR)	++/− (PR)	−/+ (leg)	−	Th3 (T2)	1 mo

Abbreviations: AE, anesthesia; bil, bilateral; B/R DF, bladder/rectal dysfunction; C, cervical vertebrae; DE, dysesthesia; f, female; HIA, high intensity area; JPS, joint position sense; L, lumbar vertebrae; lt, left; MRI, magnetic resonance imaging; m, male; No, number; PP, pinprick; PR, paresthesia; rt, right; ST, soft touch; Th, thoracic vertebrae; T1, T1-weighted image; T2, T2-weighted image; Vib, vibration; +, mild; ++, moderate; +++, severe.

Table 3. *The axial MRI findings of 4 patients*

No.	Photographing condition	MRI images and schema
5	T2-weighted image (axial)	
9	FSE T2-weighted image under fat suppression (axial)	
12	T2-weighted image (axial)	
13	T2-weighted image (axial)	

Abbreviations: FSE, fast spin echo; MRI, magnetic resonance imaging; No, number; Rt, right.

Black areas represent ischemic lesions.

well as deep sense in the dorsolateral pathway trace the same way (Fig 4, B, C).[5] Neurofibers of soft touch sense in the lateral spinothalamic tract project to the medial and

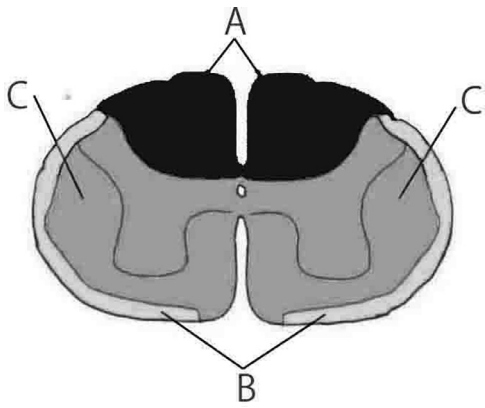


Figure 2. *Distribution of the arterial supply in the spinal cord. The posterior column is supplied with the posterior spinal cord artery (A). The lateral region is the surface layer of the anterior and lateral columns and is supplied with the coronary artery (B). The medial region includes the gray matter and almost part of the anterior and lateral columns and is supplied by the central artery (C).*

lateral parts of the thalamus (Fig 4, C).[5] In our case series, ischemia spread bilaterally in 12 patients (patients 1-12); lateral involvement was noted in 1 patient (patient 13); and 1 patient exhibited total sensory disturbances in addition to severe paraplegia (patient 1). This appearance indicated that transverse myelopathy occurred due to ischemia of the ASCA and the PSCA. In the other 11 patients excluding 1 patient (patient 8) who exhibited dysesthesia in the sensory disturbance, pain was commonly impaired, although joint position sense was preserved, which suggested that ischemia of the ASCA was the main cause of SCI. Lesions

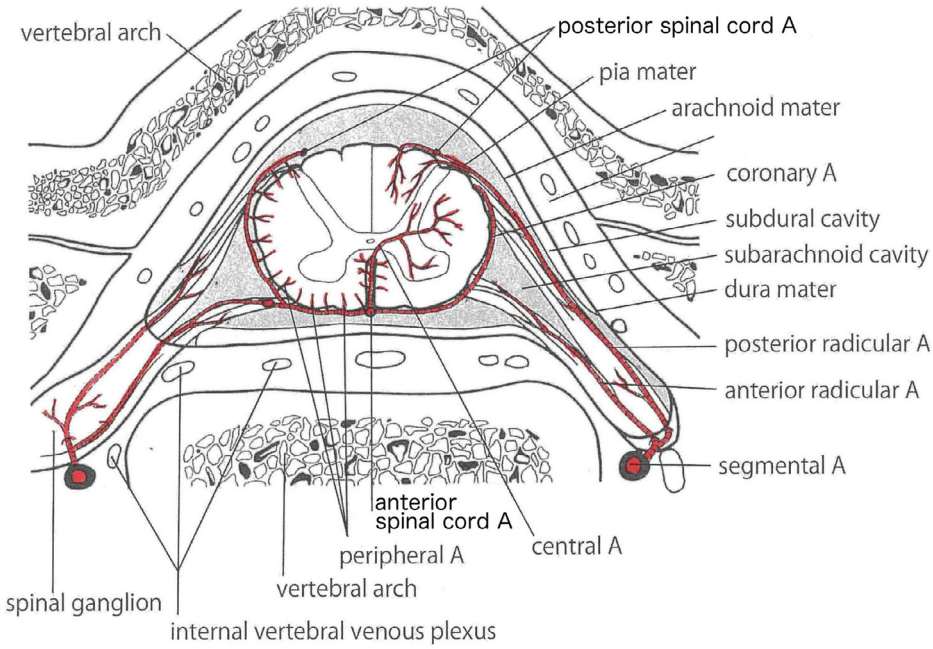


Figure 1. *Arterial supply to the spinal cord (modified from the original figure by Goto[1]). The central artery enters the spinal cord at the anterior medial fissure and supplies part of the anterior and lateral columns and central gray matter. The coronary artery anastomoses the anterior spinal artery and the posterior spinal artery. The coronary artery supplies the superficial zone of the anterior and lateral columns. The posterior spinal cord artery supplies the posterior column. Abbreviation: A, artery.*

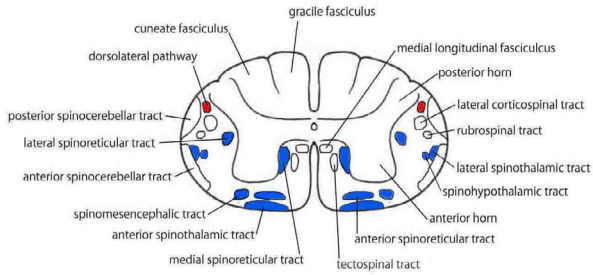


Figure 3. *Components of the anterolateral quadrant. The anterolateral quadrant is constituted of the spinothalamic, spinoreticular, spinomesencephalic, and spinohypothalamic tracts. These 4 tracts are located in the anterior and lateral columns (blue tracts). (Color version of figure is available online.)*

of 4 patients were identified in the territory of the ASCA in the axial images (patients 5, 9, 12, 13) (Table 3). The faint HIA around the lesion possibly indicated an edematous change (patient 12). Additionally, this was accompanied by impairment of vibration sense in 7 patients. There was a possibility that involvement of the dorsolateral pathway caused impairment of vibration sense because ischemia spread to the posterior part of the lateral column where the dorsolateral pathway exits.[5] On the other hand, preservation of joint position sense in these 7 patients indicated that a large proportion of the neurofibers for joint position sense are considered to be localized in the posterior column. In 4 patients without vibration sense impairment, ischemic lesions were considered not to involve the dorsolateral pathway in the posterior part of the lateral column. The neurological characteristics of lateral involvement in 1 patient were consistent with Brown-Séquard syndrome, although joint position sense was preserved. There was possible that incomplete Brown-Séquard syndrome in the lack of joint position sense impairment occurred due to ischemia in the ASCA territory.

Compared the segmental level of sensory disturbance to locations of the lesions in 7 patients whose lesions was detected, the segmental level was mapped to the part above the lesion by 1 segment in 1 patient (patient 2), the part below the lesions in 4 patients (patients 5, 6, 8, 13), and within the level of the lesions in 2 patients (patients 9, 12). The segmental level of sensory disturbance is usually mapped within the level of the lesion or at the level below the lesion in SCI.[1] This characteristic conformed to the results in our case series excluding 1 patient (patient 2).

Blood supply to the spinal cord originates from the aorta for the most part.[11-15] Dissecting aortic aneurysm and surgical procedures for aortic aneurysm are the mainstay of SCI and a cause of insufficient blood supply to the spinal cord.[11-15] In our case series, SCI in 4 patients was considered to occur by hypoperfusion in the spinal cord due to dissecting aortic aneurysm or operation for aortic aneurysm. Atherothrombosis is also 1 of a risk factor for SCI.[3, 11] In our case series, atherothrombosis was possibly associated with occurrence of SCI in the other 9 patients. In our case series, ischemia caused by dissecting aortic aneurysm or atherothrombosis possibly spread to the posterior column in the 11 patients with infarction of the territory of the ASCA. Ischemia little involved the posterior column because the 2 PSCAs supply the posterior column and connect to each other with anastomoses.[16] As a result, joint point sense was considered to be absent.

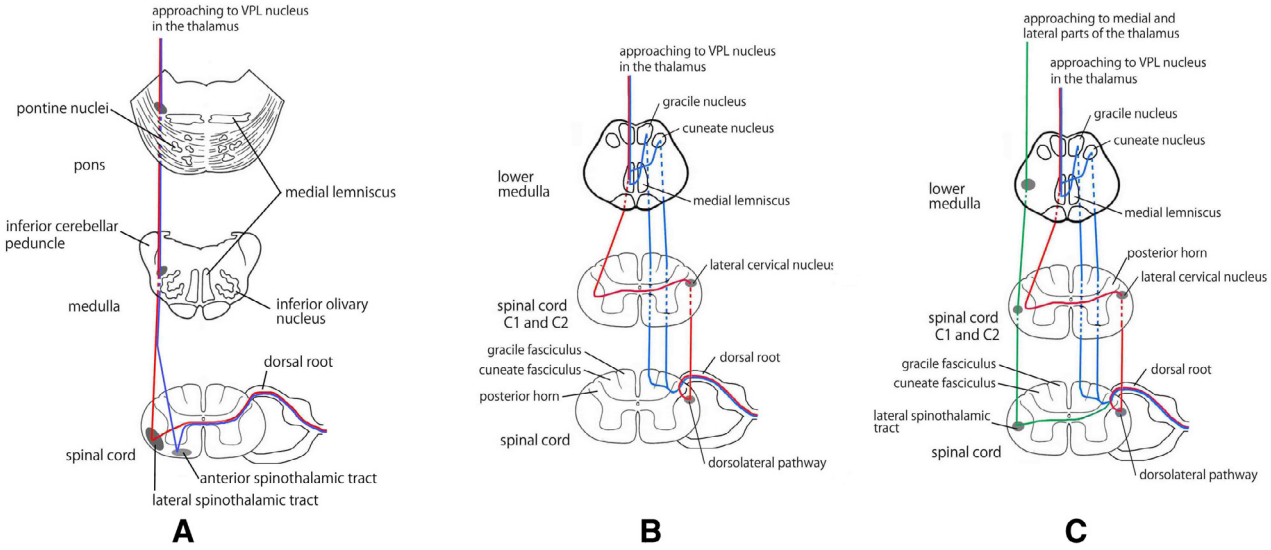


Figure 4. *Tracts of sensory information in the spinal cord (modified from the original figure by Gilman[5]). Superficial sense enters the spinal cord and decussates at the level of the entrance. Information on direct superficial sense ascends in the lateral and anterior spinothalamic tracts (A). Information on deep sense ascends in the posterior column and the dorsolateral pathway in the posterior part of the lateral column. The dorsolateral pathway reaches the lateral cervical nucleus and decussates, finally approaches the VPL nucleus (B). Information on soft touch sense enters the posterior column on the ipsilateral side. Additionally, information on soft touch sense ascends in the dorsolateral pathway on the ipsilateral side or the lateral spinothalamic tract on the contralateral side. The dorsolateral pathway reaches the lateral cervical nucleus and decussates, and finally approaches the VPL nucleus through the medial lemniscus (C). Abbreviation: VPL, ventral posterolateral.*

In conclusion, our study indicated that impairment of vibration sense can occur in SCI in the territory of the ASCA. There is a possibility that ischemia of the ASCA can spread to the posterior part of the lateral column and involve the dorsolateral pathway in this area in patients with the ASCA infarction with vibration sense impairment.

Conflict of Interest

The authors state that we have no conflict of interest (COI).

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